Exhibit E

Environomic
Environmental Medicine and Epidemiologic Consultants
110 Hickory Tavern Road
Meyersville, New Jersey 07933
Phone: 908-626-9515
Fax: 908-626-9517

Methyl Tertiary Butyl Ether and Human Health Effects

Expert Report in the Matter Concerning:
City of New York v.
Amerada Hess Corp., et al.

Prepared by

Sandra N. Mohr, MD, MPH

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Sandra N. Mohr, M.D. M.P.H. 3/09/09

DEFENDANT'S EXHIBIT
D-12466

COMMENTARY ON PLAINTIFFS' EXPERT REPORT

I have read the report by Dr. Kathleen Burns. Please note that on page ten of her report, Dr. Burns states that she "adapted summary tables" from the "Toxicological Profile of [sic] MTBE" by the CDC. In actuality, the ATSDR "Toxicological Profile for MTBE" contains no such table. It does contain table 2.1 on page 11 of that document which does list the two human inhalation studies by Prah and Cain summarized earlier in this report. Not only does the ATSDR table not list "eye irritation" as a consequence of the MTBE exposure in these two studies, the studies themselves do not report any eye irritation or changes in tear film breakup time. (Similarly there were no ocular symptoms from exposure to more than 20 times this level in humans in the Nihlen study.)

Additionally, the ATSDR Table 2.1 on page 11 lists the levels from those two studies of 1.7 ppm (1700 ppb) and 1.39 ppm (1390 ppb) as "No Observable Adverse Effect Levels" in humans. This is contrary to Dr. Burns assertion on page 5 of her report that there is "no credible or proven 'safe' level of MTBE exposure" and she leaves out this part of the ATSDR table in her own "adapted" one. She also includes a row in her "adapted" table regarding gallbladder treatment and does acknowledge that this row is not in the ATSDR report. In the row she lists numerous treatment effects, but does not provide any references for her health effects assertions. As a nonclinician, it is unclear whether Dr. Burns has the expertise to discern the difference between liver abnormalities due to the gallstones and those due to the treatment. The actual experience with using MTBE as a treatment is summarized and the actual references cited on page 7 of this report.

On page 13 of Dr. Burn's report she states that, "it is impossible to predict how individuals will respond at specific exposure levels." This statement is contrary to the science of toxicology. The concept of specific exposure level, or dose, is a basic tenet in science and well recognized by society. We do not expect the same behavior (toxicity) from a person who drinks an 8 ounce glass of an alcoholic beverage as we do from one who drinks one drop of alcohol in an 8 ounce glass of water, nor do we assign as a society the same consequences to those behaviors. It is well recognized that acetaminophen (sold as Tylenol and other brands) causes liver failure and death in humans, but not at the doses recommended for fever and pain relief. As a society we can and do regulate chemicals at levels that we believe are safe for even the most sensitive members of the population.

Dr. Burns relies solely on mutagenicity assays and animal studies and fails to review the published human literature on MTBE exposure. Furthermore, she is very selective of which mutagenicity studies she chooses to rely upon. According to the USEPA, which reviewed all available mutagenicity studies at the time, "with one exception, this chemical [MTBE] has not exhibited genetic toxicity in a variety of in vitro and in vivo mammalian and non-mammalian test systems." The single exception was a mouse lymphoma assay which was thought to be positive due to *in vitro* and not *in vivo* metabolism and therefore of limited importance. The USEPA summarized that the weight of evidence "indicated that MTBE is not mutagenic" (EPA 1997). The USEPA document was "peer reviewed both internally in the Agency and externally by experts in the field

before its release to the public" (EPA 1997). More recently, twenty-five genotoxicity assays were reviewed with the author finding, "The data indicative of genotoxicity are very weak, with none of the studies indicating significant activity having been independently verified, except for the mutagenicity in mouse lymphoma cells" (McGregor 2006). Dr. Burns relies upon Du et al. citing that MTBE causes DNA adducts. However, this study has been criticized in that the way it was conducted, it was impossible for the authors to know whether the tagged carbon on the DNA came from a DNA adduct or from the metabolism of MTBE to formaldehyde and from the 1-carbon pool into normal incorporation into DNA (Swenberg 2008).

Dr. Burns condemns "industry" for misinterpreting studies looking at MTBE exposures and developmental outcomes and stating these studies were referred to as "showing that MTBE did not cause any particular hazards to pregnant women or children" (page 25). This statement is misleading, because in reality none of these studies deal with pregnant women or children at all as they are all animal studies. Like tumor registries, many states have birth defects registries. The New York State Congenital Malformations Registry is one of 34, which submits data to the National Birth Defects Prevention Network. The data in the registry undergoes consistent surveillance for changes in rates and trends (New York State Department of Health 2004). Despite MTBE being in use for 30 years in gasoline, there have been no reported birth defects clusters around MTBE spill sites, nor have there been any reported clusters around manufacturing sites.

Dr. Burns opines that MTBE is a carcinogen. This statement is very misleading as there is no reliable scientific basis to conclude that MTBE or its metabolites may be carcinogenic based on the levels of MTBE to which humans may be exposed in drinking water. She fails to acknowledge that other panels of reputable (and non "industry" scientists) have reviewed the same data as she has and neither the National Toxicology Program at the National Institutes of Health, IARC nor the EU, have voted to list MTBE as a human carcinogen. She notes that MTBE metabolites, TBA and formaldehyde, are also carcinogenic. TBA, used in the manufacture of perfumes, has been studied in some animal assays but is not classified as a human carcinogen under USEPA. While high doses of formaldehyde are carcinogenic, a small amount of formaldehyde formation is a normal part of the metabolism of foods in the human body and is essential in de novo purine synthesis and amino acid synthesis (nucleic acid and protein manufacture) as noted by Dr. Swenberg.

Dr. Burns opines that MCL levels for MTBE in water vary from state to state but does not address the difference between a MCL for protecting health and a guideline that is set even lower for organoleptic reasons. The USEPA did originally propose to issue an MCL for MTBE at 70 ug/L as a health-based standard. However, because of taste and odor studies, some conducted in California, it became clear that this standard, while protective for health, did not address issues of water taste and odor. USEPA has not issued a primary MCL to date, and instead has issued secondary guidelines based on organoleptic (odor and taste) properties at a range of 20-40 ug/L (20-40 ppb) that would help ensure consumer acceptance and were even more protective. They note that at 20 ug/L, the margin of exposure (or safety) is approximately 40 thousand times less than the range of cancer effects seen in animals at high doses and would be twice that (80